

=> d his

(FILE 'HOME' ENTERED AT 12:49:30 ON 10 NOV 2007)

FILE 'REGISTRY' ENTERED AT 12:49:40 ON 10 NOV 2007

L1 STRUCTURE UPLOADED
L2 0 S L1 SSS SAM
L3 5 S L1 SSS FULL

FILE 'CAPLUS, MEDLINE' ENTERED AT 12:51:21 ON 10 NOV 2007

L4 169 S L3
L5 8 S L4 AND PROMOTE?
L6 161 S L4 NOT L5
L7 2 S L6 AND HYALURONIC ACID?
L8 159 S L6 NOT L7
L9 1 S L6 AND SYNTHASE GENE?
L10 158 S L8 NOT L9
L11 17 S L10 AND EXPRESSION
L12 4 S L11 AND GENE?
L13 13 S L11 NOT L12
L14 141 S L10 NOT L11
L15 0 S L14 AND ?OSTEOARTHRITIS
L16 0 S L14 AND ?OSTEOARTH?
L17 0 S L14 AND ?OSTEO?
L18 0 S L14 AND ?ARTHRITIS
L19 3 S L14 AND ?AGING
L20 138 S L14 NOT L19
L21 0 S L20 AND HYALURONAN
L22 0 S L20 AND HYALURONATE
L23 0 S L22 AND SYNTHETASE?
L24 0 S L22 AND SYNTHASE
L25 0 S L20 AND SYNTHETASE?
L26 1 S L20 AND SYNTHASE
L27 1 S L20 AND HYALUR?
L28 0 S L20 AND GLYCOAMINOGLYCAN?
L29 0 S L20 AND POLYSACCHARIDE?
L30 2 S L20 AND COSMETIC?
L31 136 S L20 NOT L30
L32 1 S L31 AND SKIN?

=> d his

(FILE 'HOME' ENTERED AT 12:49:30 ON 10 NOV 2007)

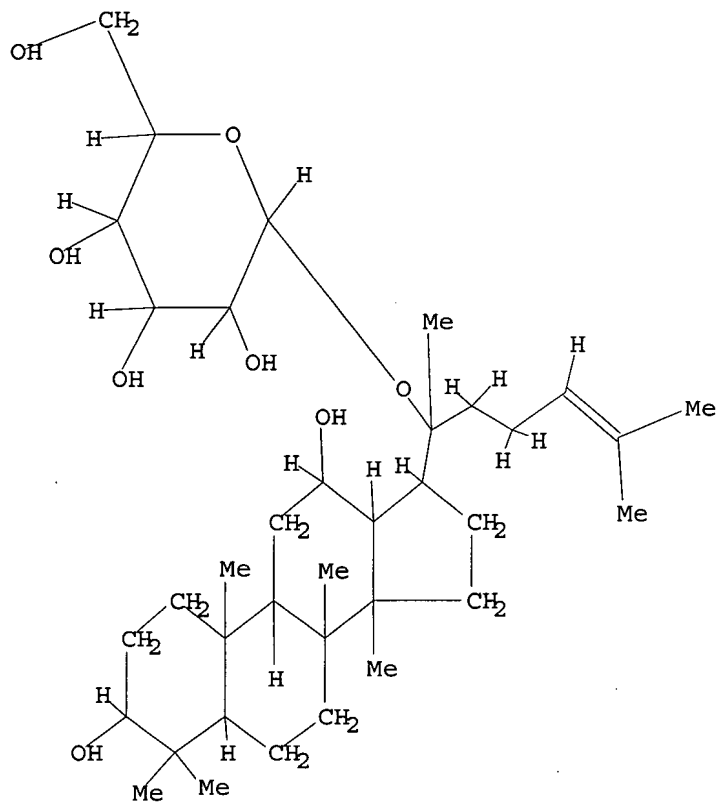
FILE 'REGISTRY' ENTERED AT 12:49:40 ON 10 NOV 2007

L1 STRUCTURE UPLOADED
L2 0 S L1 SSS SAM
L3 5 S L1 SSS FULL

FILE 'CAPLUS, MEDLINE' ENTERED AT 12:51:21 ON 10 NOV 2007

L4 169 S L3
L5 8 S L4 AND PROMOTE?
L6 161 S L4 NOT L5
L7 2 S L6 AND HYALURONIC ACID?
L8 159 S L6 NOT L7
L9 1 S L6 AND SYNTHASE GENE?
L10 158 S L8 NOT L9
L11 17 S L10 AND EXPRESSION
L12 4 S L11 AND GENE?
L13 13 S L11 NOT L12
L14 141 S L10 NOT L11
L15 0 S L14 AND ?OSTEOARTHRITIS
L16 0 S L14 AND ?OSTEOARTH?
L17 0 S L14 AND ?OSTEO?
L18 0 S L14 AND ?ARTHRITIS
L19 3 S L14 AND ?AGING
L20 138 S L14 NOT L19
L21 0 S L20 AND HYALURONAN
L22 0 S L20 AND HYALURONATE
L23 0 S L22 AND SYNTHETASE?
L24 0 S L22 AND SYNTHASE
L25 0 S L20 AND SYNTHETASE?
L26 1 S L20 AND SYNTHASE
L27 1 S L20 AND HYALUR?
L28 0 S L20 AND GLYCOAMINOGLYCAN?
L29 0 S L20 AND POLYSACCHARIDE?
L30 2 S L20 AND COSMETIC?
L31 136 S L20 NOT L30
L32 1 S L31 AND SKIN?

=> d L1
L1 HAS NO ANSWERS
L1 STR



Structure attributes must be viewed using STN Express query preparation.

ANSWER 1 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:1319220 CAPLUS
DOCUMENT NUMBER: 144:64043
TITLE: Ginseng saponin metabolite suppresses phorbol
ester-induced matrix metalloproteinase-9 expression
through inhibition of activator protein-1 and
mitogen-activated protein kinase signaling pathways in
human astroglioma cells
AUTHOR(S): Jung, Soo-Hyun; Woo, Moon-Sook; Kim, So-Young; Kim,
Won-Ki; Hyun, Jin-Won; Kim, Eun-Jin; Kim, Dong-Hyun;
Kim, Hee-Sun
CORPORATE SOURCE: Department of Neuroscience, Ewha Institute of
Neuroscience, College of Medicine, Ewha Woman's
University, Seoul, S. Korea
SOURCE: International Journal of Cancer (2005), Volume Date
2006, 118(2), 490-497
CODEN: IJCNAW; ISSN: 0020-7136
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

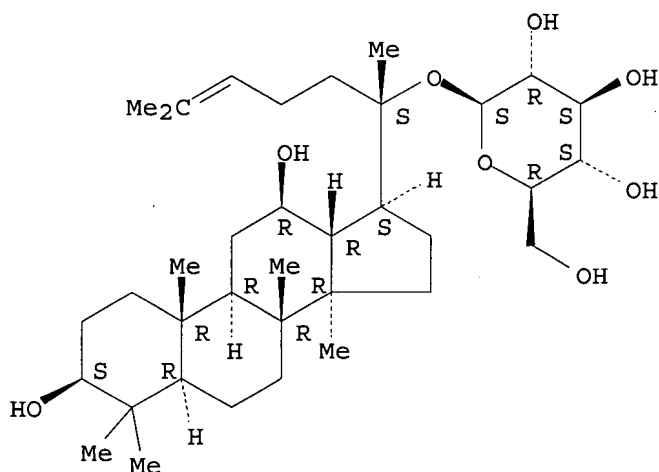
AB Aberrant expression of matrix metalloproteinase-9 (MMP-9) is implicated in
the process of invasion and angiogenesis of malignant tumors as well as in
inflammatory diseases of the CNS. Therefore, the development of compds.
that can inhibit or suppress MMP-9 is required to treat brain tumors. We
investigated the effects of a ginseng saponin metabolite, compound K
(20-O-(β -D-glucopyranosyl)-20(S)-protopanaxadiol), on MMP-9
expression in human astroglioma cells. Compound K significantly inhibited
the secretion and protein expression of MMP-9 induced by PMA. The
inhibitory effect of compound K on MMP-9 expression correlated with
decreased MMP-9 mRNA levels and suppression of MMP-9 promoter
activity. The compound K-mediated inhibition of MMP-9 gene expression
appears to occur via AP-1 because its DNA-binding and transcriptional
activities were suppressed by the agent. Furthermore, compound K
significantly repressed the PMA-mediated activation of p38 MAPK, ERK and
JNK, which are upstream modulators of AP-1. Finally, compound K inhibited
the in vitro invasiveness of glioma cells. Therefore, inhibition of MMP-9
expression by compound K might have therapeutic potential for controlling
the growth and invasiveness of brain tumors.

IT 39262-14-1, IH-901
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU
(Therapeutic use); BIOL (Biological study); USES (Uses)
(ginseng saponin metabolite effect on matrix metalloproteinase-9
expression through inhibition of activator protein-1 and
mitogen-activated protein kinase signaling pathways in human
astroglioma cells)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-
yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:64871 CAPLUS

DOCUMENT NUMBER: 142:309370

TITLE: Antitumor promotional effects of a novel intestinal bacterial metabolite (IH-901) derived from the protopanaxadiol-type ginsenosides in mouse skin
 AUTHOR(S): Lee, Ji-Yoon; Shin, Jun-Wan; Chun, Kyung-Soo; Park, Kwang-Kyun; Chung, Won-Yoon; Bang, Yung-Jue; Sung, Jong-Hwan; Surh, Young-Joon

CORPORATE SOURCE: National Research Laboratory of Molecular Carcinogenesis and Chemoprevention, College of Pharmacy, Seoul National University, Seoul, 151-742, S. Korea

SOURCE: Carcinogenesis (2005), 26(2), 359-367

CODEN: CRNGDP; ISSN: 0143-3334

PUBLISHER: Oxford University Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Epidemiol. studies have demonstrated that ginseng intake decreases the risk of cancer. Ginseng saponins (ginsenosides) have been regarded as principal components responsible for the majority of pharmacol. activities exerted by ginseng. IH-901 [20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol], an intestinal bacterial metabolite derived from protopanaxadiol-type saponins of Panax ginseng C.A. Meyer, has been reported to possess antitumor effects, including inhibition of invasion, metastasis and angiogenesis and induction of tumor cell apoptosis. Tumor promotion often accompanies an elevated ornithine decarboxylase (ODC) activity, acute inflammation and induction of cyclooxygenase-2 (COX-2) activity. Here the authors examined the effects of IH-901 on tumor promotion and related mol. events in mouse skin in vivo. Mouse ear edema induced by the prototype tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) was repressed by IH-901 pretreatment in a dose-dependent manner. Topical application of IH-901 onto shaven backs of female ICR mice led to the inhibition of TPA-induced expression of COX-2 and production of prostaglandin E2. The eukaryotic transcription factor NF- κ B has been involved in intracellular signaling pathways associated with inflammation and carcinogenesis. IH-901 pretreatment inhibited TPA-induced epidermal NF- κ B DNA binding in mouse skin, which appeared to be mediated by blocking phosphorylation and subsequent degradation of I κ B α . In an attempt to elucidate the mol. mechanisms by which IH-901 inactivates NF- κ B, its effects on activation of upstream signaling kinases were explored. IH-901 also

inhibited the activation of ERK1/2 and Akt signaling. When IH-901 was treated topically prior to TPA, expression and activity of ODC were inhibited dose-dependently. In addition, IH-901 given prior to each topical dose of TPA markedly lowered the number of papillomas in mouse skin induced by 7,12-dimethylbenz[a]anthracene. Taken together, these findings suggest that IH-901 exerts anti-inflammatory effects by inhibiting TPA-induced COX-2 expression, which may contribute to its antitumor-promoting effects on mouse skin carcinogenesis.

IT 39262-14-1, IH-901

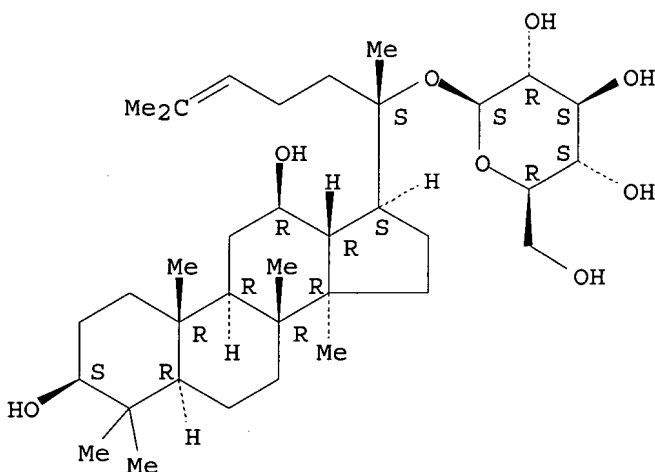
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(antitumor effects of an intestinal bacterial metabolite (IH-901) derived from the protopanaxadiol-type ginsenosides in mouse skin)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:566639 CAPLUS

DOCUMENT NUMBER: 141:117187

TITLE: 20-O- β -D-Glucopyranosyl-20(S)-protopanaxadiol for promotion of the production of hyaluronic acid

INVENTOR(S): Kim, Su-Jong; Kang, Byung-Young; Cho, Si-Young; Chang, Hui-Kyoung; Sung, Dae-Seok; Yeom, Myeong-Hoon; Woe, Kwang-Sik; Kim, Duck-Hee; Kim, Han-Kon; Sim, Young-Chul; Kang, Hak-Hee; Lee, Yong-Sung

PATENT ASSIGNEE(S): Amorepacific Corporation, S. Korea

SOURCE: PCT Int. Appl., 29 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004058796	A1	20040715	WO 2003-KR1889	20030916
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KZ, LC, LK, LR,			

LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
 PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
 TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
 RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
 KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
 FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
 BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

KR 2004057339 A 20040702 KR 2002-84036 20021226
 AU 2003261657 A1 20040722 AU 2003-261657 20030916
 EP 1575982 A1 20050921 EP 2003-813984 20030916

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
 IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

CN 1717414 A 20060104 CN 2003-825714 20030916
 JP 2006515303 T 20060525 JP 2004-562980 20030916
 US 2006160752 A1 20060720 US 2005-539011 20051230

PRIORITY APPLN. INFO.: KR 2002-84036 A 20021226
 WO 2003-KR1889 W 20030916

AB The invention provides a promoter containing ginsenoside compound
 20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol (compound K; preparation
 described) for the production of hyaluronic acid. Compound K is a chief
 metabolite of ginseng saponin and is used to increase the expression of
 hyaluronic acid synthase gene in human cells and thereby to
 promote the production of hyaluronic acid. Also disclosed is an
 anti-aging agent containing the above compound as an active ingredient.

IT 39262-14-1

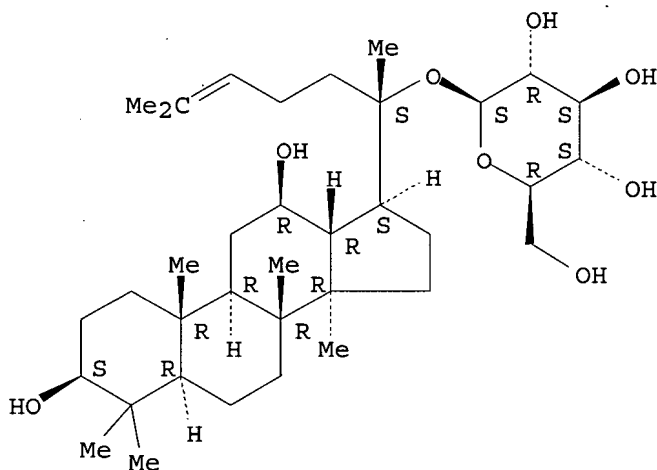
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
 (Biological study); USES (Uses)

(glucopyranosyl protopanaxadiol for promotion of production of hyaluronic
 acid)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-
 yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L5 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:558607 CAPLUS

DOCUMENT NUMBER: 141:218566

TITLE: Dendritic cells maturation promoted by M1
 and M4, end products of steroidal ginseng saponins
 metabolized in digestive tracts, drive a potent Th1
 polarization

AUTHOR(S): Takei, Masao; Tachikawa, Eiichi; Hasegawa, Hideo; Lee,
 Je-Jung

CORPORATE SOURCE: Division of Cellular Allergology, Research Center
Borstel, Parkallee, D-23845, Germany
SOURCE: Biochemical Pharmacology (2004), 68(3), 441-452
CODEN: BCPA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Ginseng is a medicinal herb widely used in Asian countries, and many of its pharmacol. actions are attributed to the ginsenosides. Dendritic cells (DCs) play a pivotal in the initiation of T-cell-mediated immune responses, making them an attractive cellular adjuvant for use in cancer vaccines. In this study, the authors investigated whether M1 and M4, end products of steroidal ginseng saponins metabolized in digestive tracts, can drive DCs maturation from human monocytes in vitro. Human monocytes were cultured with GM-CSF and IL-4 for 6 days, followed by another 2 days in the presence of M1, M4 or TNF- α as a maturation stimulus. Stimulation with 20 μ M of M1 or M4 increased expression level of CD80, CD83 and CD86 as expressed by mean fluorescence intensity (MFI) and decreased endocytic activity. M4-primed mature DCs also displayed enhanced T cells stimulatory capacity in a MLR, as measured by T cell proliferation. Mature DCs differentiated with M1 or M4 induced the differentiation of naive T cells towards a helper T cell type 1 (Th1) response at DC/T (1:5) cells ratio depending on IL-12 secretion. In CTL assay, the production of IFN- γ and 51Cr release on M4-primed mature DCs was more augmented than of immature DCs or TNF- α -primed mature DCs. These results suggest that M4 may be used on DC-based vaccines for cancer immunotherapy.

IT 39262-14-1, Protopanaxadiol 20-O-glucoside

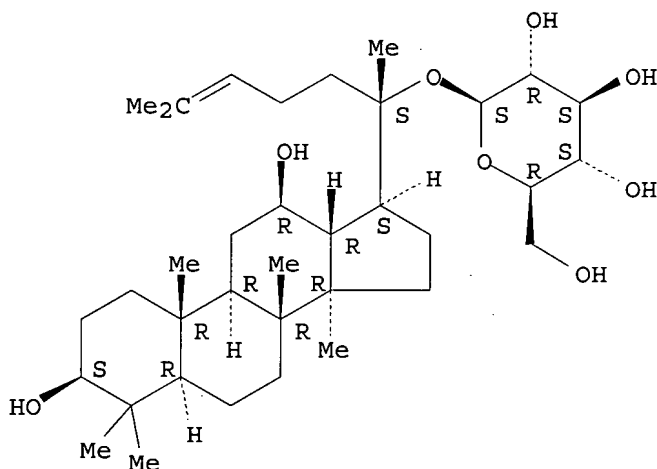
RL: PAC (Pharmacological activity); BIOL (Biological study)

(dendritic cells maturation promoted by M1 and M4, end products of steroidal ginseng saponins metabolized in digestive tracts, drive potent Th1 polarization)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:151887 CAPLUS

DOCUMENT NUMBER: 137:179207

TITLE: Metabolic activation of ginsenoside against cancer:

Intestinal bacterial deglycosylation and hepatic fatty-acid esterification

AUTHOR(S): Hasegawa, Hideo

CORPORATE SOURCE: Itto Institute of Life Science Research, Happy World Inc., Fuchu, Tokyo, 183-0011, Japan

SOURCE: Wakan Iyakugaku Zasshi (2001), 18(6), 217-228
CODEN: WIZAEL; ISSN: 1340-6302

PUBLISHER: Wakan Iyaku Gakkai

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Despite the commonly accepted concept that ginsenoside is responsible for the pharmacol. effects of ginseng (Panax ginseng), there were few systematic studies of the metabolic activation of ginsenoside in the body after oral administration of ginseng. Although, ginsenoside was known to be metabolized by intestinal bacteria, it was obscure whether or not the metabolites participate ginseng actions. For this reason, we analyzed the interrelationship between ginsenoside, formation of intestinal bacterial metabolites, pharmacokinetics of metabolites and preventive actions against cancers. Orally administered ginsenoside passed through the stomach and small intestine without decomposition by either gastric juice or liver enzymes into the large intestine, where ginsenoside was deglycosylated by colonic bacteria. 20(S)-Protopanaxadiol monoglucoside (M1) was a major metabolite of protopanaxadiol-type ginsenoside. Following absorption of M1 in the blood, it transferred into the liver with a high degree of selectivity. M1 directly exerted antitumor effects such as induction of apoptosis and cell cycle arrest and inhibition of tumor-induced neovascularization; however, much higher doses of M1 were toxic to the host. Thus, most of the absorbed M1 was excreted rapidly as bile, and some residual M1 (approx. 25% of the dose) was esterified with fatty acids. The esterified form (EM1) was sustained in the liver longer than M1. Thus, the in vivo antitumor activity paralleled with the pharmacokinetic behavior. EM1 did not directly affect tumor growth in vitro, whereas it stimulated lymphocytes to become cytotoxic to tumor cells. The esterification of M1 with fatty acids potentiated the antitumor activity of M1 through delay of the clearance and through immunostimulation. These findings have given a clue to the mechanism underlying expression of ginseng actions in the body.

IT 39262-14-1 39262-14-1D, fatty acid esters

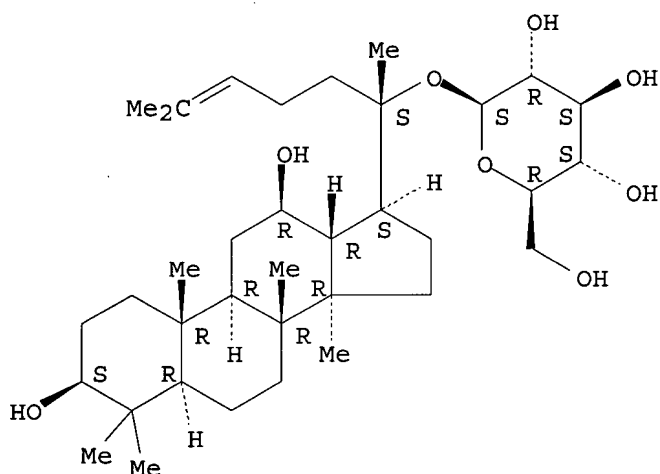
RL: BSU (Biological study, unclassified); DMA (Drug mechanism of action); PAC (Pharmacological activity); PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(ginsenoside metabolite; metabolic activation of ginsenoside against cancer by intestinal bacterial deglycosylation and hepatic fatty-acid esterification)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

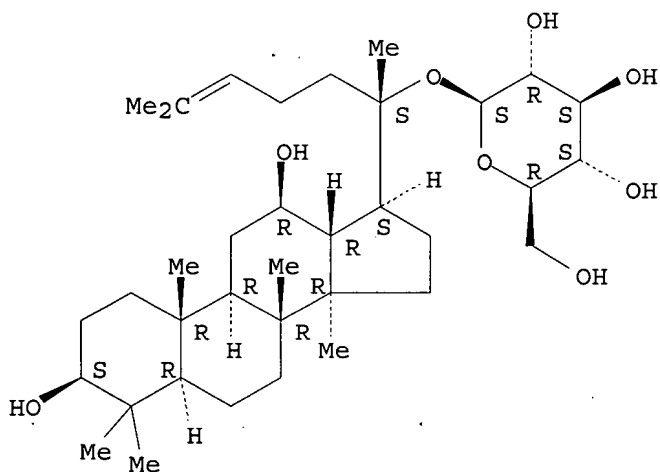
Absolute stereochemistry. Rotation (+).



RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2001:115735 CAPLUS

DOCUMENT NUMBER: 135:116708

TITLE: Oleoyl triterpene glycoside biotransformed from ginseng suppresses growth and metastasis of murine B16-F10 melanoma via immunostimulation

AUTHOR(S): Hasegawa, Hideo; Saiki, Ikuo

CORPORATE SOURCE: Department of Pathogenic Biochemistry, Toyama Medical and Pharmaceutical University, Japan

SOURCE: Wakan Iyaku Gaku Zasshi (2000), 17(5), 186-193
CODEN: WIZAEL; ISSN: 1340-6302

PUBLISHER: Wakan Iyaku Gakkai

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of 20(S)-protopanaxadiol 20-O- β -D-glucopyranoside (M1) and 3-O-oleoyl M1 (OM1) on the growth and metastasis of murine B16-F10 melanoma cells were examined in C57BL/6 mice. A single co-injection of M1

(5 mg/kg) with B16-F10 cells into the liver inhibited tumor growth at the inoculation site by 23% (not significant compared to untreated control). In contrast, the same dosage of OM1 caused a 2.6-fold suppression of tumor growth, compared with M1 treatment. Concerning the pharmacokinetics, both M1 and OM1 were selectively taken up into the liver soon after i.v. administration (30 mg/kg). Thereafter, M1 was cleared immediately from the liver; however, OM1 was retained in the liver at a level of more than 25% of the administered dose for 24 h after administration. Thus, the antitumor activity paralleled the pharmacokinetic behavior. Moreover, three consecutive i.v. administrations of OM1 (30 mg/kg) inhibited the liver metastasis produced by intrasplenic inoculation of B16-F10 cells by 95%. OM1 did not directly affect tumor growth in vitro, whereas it promoted tumor cell lysis by lymphocytes, particularly non-adherent splenocytes, in a concentration-dependent manner. These results suggest that fatty acid esterification of M1 potentiates the antitumor activity of the parental M1 through delay of the clearance and through immunostimulation.

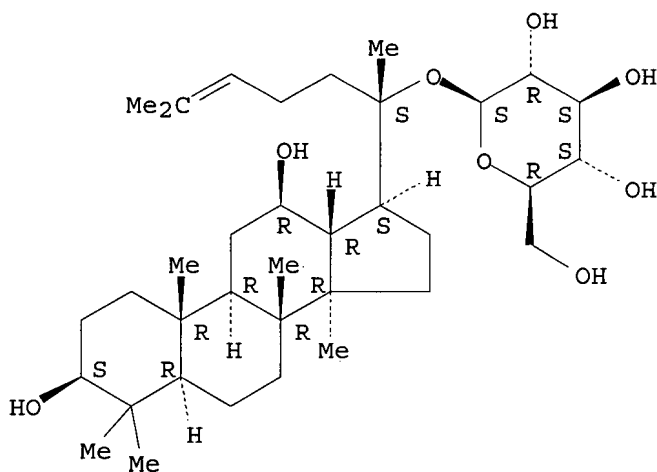
IT 39262-14-1

RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses) (oleoyl triterpene glycoside biotransformed from ginseng suppresses growth and metastasis of murine B16-F10 melanoma via immunostimulation)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2000:655518 CAPLUS

DOCUMENT NUMBER: 134:95201

TITLE: An intestinal bacterial metabolite (M1) of ginseng protopanaxadiol saponins inhibits tumor-induced neovascularization

AUTHOR(S): Suda, Kazuhito; Murakami, Koji; Murata, Jun; Hasegawa, Hideo; Saiki, Ikuro

CORPORATE SOURCE: Department of Pathogenic Biochemistry, Institute of Natural Medicine, Toyama Medical and Pharmaceutical University, Toyama, Japan

SOURCE: Wakan Iyakugaku Zasshi (2000), 17(4), 144-150
CODEN: WIZAEL; ISSN: 1340-6302

PUBLISHER: Wakan Iyaku Gakkai

DOCUMENT TYPE: Journal
LANGUAGE: English

AB An intestinal bacterial metabolite (M1) of ginsenoside Rb1 [structure given in original], when administered orally to mice, inhibited the growth of implanted tumor and its intrahepatic metastasis following implantation of a small fragment of colon 26-L5 tumor into the liver. These findings indicate that M1 was effective in inhibiting the growth and metastasis of colon 26-L5 cells, in addition to inhibiting the lung metastasis of B16-BL6 melanoma cells, as reported previously. The conditioned medium from the growth of colon 26-L5 cells (CM-L5) induced in vitro tube formation of hepatic sinusoidal endothelial (HSE) cells on Matrigel-coated substrates, considered to be an important step in the processes of tumor angiogenesis. This activity of CM-L5 was concentration-dependently abrogated

by noncytotoxic concns. of M1. Similarly, M1 concentration-dependently eliminated the ability of CM-L5 to promote the migration of HSE cells.

M1-induced inhibition of tumor growth and intrahepatic metastasis may be partly related to the suppression of tumor angiogenic responses, including capillary tube formation and migration of HSE cells.

IT 39262-14-1

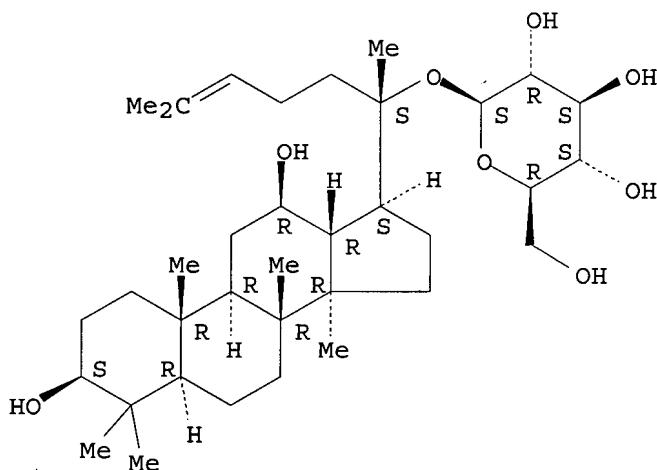
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(intestinal bacterial metabolite of ginsenoside Rb1 inhibition of colon cancer-induced neovascularization)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1985:416390 CAPLUS

DOCUMENT NUMBER: 103:16390

TITLE: Potentiation of nerve growth factor-mediated nerve fiber production in organ cultures of chicken embryonic ganglia by ginseng saponins: structure-activity relationship

AUTHOR(S): Takemoto, Yumi; Ueyama, Takashi; Saito, Hiroshi; Horio, Shuhei; Sanada, Shuichi; Shoji, Junzo; Yahara, Shoji; Tanaka, Osamu; Shibata, Shoji

CORPORATE SOURCE: Inst. Med. Dent. Eng., Tokyo Med. Dent. Univ., Tokyo,

101, Japan
SOURCE: Chemical & Pharmaceutical Bulletin (1984), 32(8),
3128-33
CODEN: CPBTAL; ISSN: 0009-2363
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Potentiation of the nerve growth factor (NGF) [9061-61-4]-mediated nerve fiber production in organ cultures of chicken embryonic dorsal root ganglia (DRG) and lumbar sympathetic ganglia (SymG) by saponins isolated from *Panax ginseng* and *P. japonicum* and related compds. was studied to elucidate structure-activity relations. *Panax* Saponins and related compds. did not promote nerve fiber production but some 20(S)-protopanaxadiol glycosides having glucose units in their 2 sugar moieties potentiated the effect of NGF. Removal of glucose or introduction of a hydroxy group into the side chain of ginsenoside Rd [52705-93-8] reduced the activity. Little difference was observed in the potentiation of the NGF effect by the saponins in organ cultures of DRG and SymG.

IT 39262-14-1

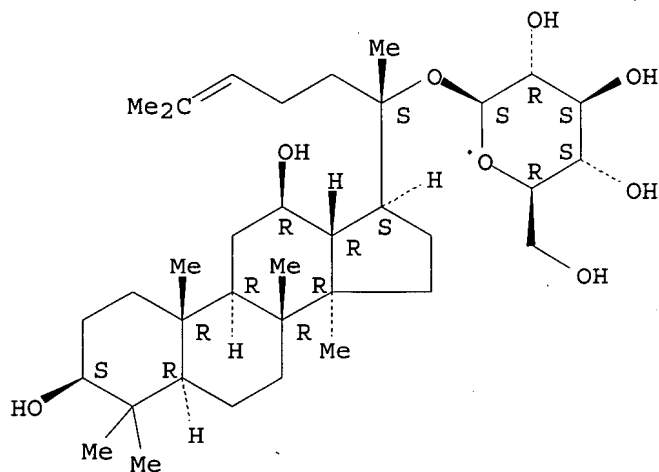
RL: BIOL (Biological study)

(nerve growth factor-mediated nerve fiber production potentiation by, in ganglion culture, structure in relation to)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L7 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2007:336655 CAPLUS

DOCUMENT NUMBER: 147:133753

TITLE: Effect of Compound K on chronic hepatic injury induced by carbon tetrachloride (CCl₄) in rats

AUTHOR(S): Zhang, Leiming; Fu, Fenghua; Wang, Tian; Han, Bing; Zhu, Mei

CORPORATE SOURCE: School of Pharmacy, Yantai University, Yantai, Shandong Province, 264005, Peop. Rep. China

SOURCE: Shizhen Guoyi Guoyao (2006), 17(1), 38-39

CODEN: SGGHAL; ISSN: 1008-0805

PUBLISHER: Shizhen Guoyi Guoyao Bianweihui

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB In male Wistar rats, hepatic chronic injury was induced by s.c. administration of CCl₄ twice a week for six weeks. Then rats were given Compound K at doses of 0.3, 1 or 3 mg/kg resp. for a further four weeks. At the end, the biochem. parameters associated with hepatic injury were determined,

and the histopathol. of liver was observed Compared with the model group, activities of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were significantly reduced by Compound K at dose of 0.3 mg/kg, accompanied with amount of superoxide dismutase (SOD) increased markedly and that of malondialdehyde (MDA) reduced obviously, while no significant changes in the amts. of hyaluronic acid (HA) and procollagen III (PCIII) or histopathol. were observed Compound K at low dose could protect liver from injury induced by CCl₄, which may be associated with its antioxidn. effect.

IT 39262-14-1, Ginsenoside "K"

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL

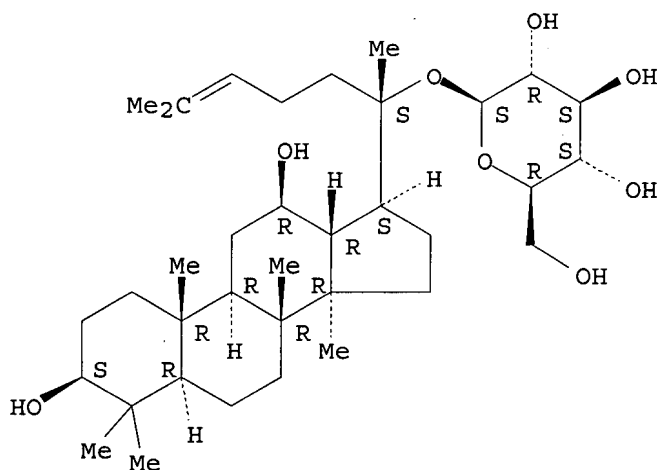
(Biological study); USES (Uses)

(effect of Compound K on chronic hepatic injury induced by carbon tetrachloride (CCl₄) in rats)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L7 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2006:699710 CAPLUS

DOCUMENT NUMBER: 145:159858

TITLE: Application of ginsenoside C-K in preparing drugs for

treating and preventing hepatic fibrosis

INVENTOR(S): Fu, Fenghua; Li, Guisheng; Lei, Qingzhong; Liu, Ke; Zhang, Leiming; Wang, Tian

PATENT ASSIGNEE(S): Shandong Luye Natural Medical Research Developing Co., Ltd., Peop. Rep. China

SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 9 pp.
CODEN: CNXXEV

DOCUMENT TYPE: Patent

LANGUAGE: Chinese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1679600	A	20051012	CN 2005-10054634	20050307
PRIORITY APPLN. INFO.:			CN 2004-10023663	A 20040309

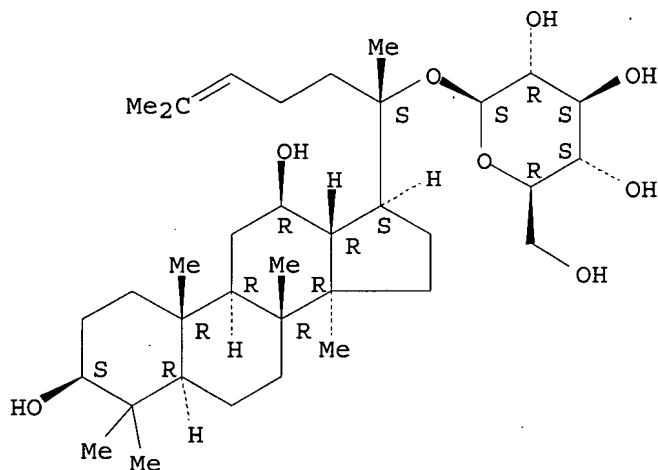
AB The invention provides in details the application of ginsenoside C-K in preparing drugs for treating and preventing hepatic fibrosis caused by infection and chemical factors. The drug containing ginsenoside C-K can be tablet, capsule, dripping pill, injection, etc.

IT 39262-14-1
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (application of ginsenoside C-K in preparing drugs for treating and preventing hepatic fibrosis)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



L9 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:216268 CAPLUS

DOCUMENT NUMBER: 140:368605

TITLE: Compound K induces expression of hyaluronan synthase 2 gene in transformed human keratinocytes and increases hyaluronan in hairless mouse skin

AUTHOR(S): Kim, Sujong; Kang, Byung Young; Cho, Si Yong; Sung, Dae Suk; Chang, Hui Kyung; Yeom, Myung Hun; Kim, Duk Hee; Sim, Young Chul; Lee, Yong Sung

CORPORATE SOURCE: R&D Center, Yongin-si Amore-Pacific Corporation, Kiheung-eup, Yongin-si, Kyounggi-do, 449-729, S. Korea

SOURCE: Biochemical and Biophysical Research Communications (2004), 316(2), 348-355

CODEN: BBRCA9; ISSN: 0006-291X

PUBLISHER: Elsevier Science

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Ginsenosides, the major active ingredients of ginseng, have a variety of biomedical efficacies such as anti-aging, anti-oxidation, and anti-inflammatory activities. To understand the effects of compound K (20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol), one of the major metabolites of ginsenosides, on the skin, we assessed the expression levels of about 100 transcripts in compound K-treated HaCaT cells using cDNA microarray anal. One gene up-regulated by compound K was hyaluronan synthase2 (HAS2). Semi-quant. RT-PCR showed that compound K increased HAS2 mRNA in time- and dose-dependent manners. ELISA and immunocytochem. using hyaluronan (HA)-binding protein showed that compound K effectively increased HA production in HaCaT cells. Finally, treatment of compound K on hairless mouse skin increased the amount of HA in the epidermis and papillary dermis. Our study suggests that topical application of compound K might prevent or improve the deteriorations, such as xerosis and wrinkles, partly ascribed to the age-dependent decrease of the HA content in human skin.

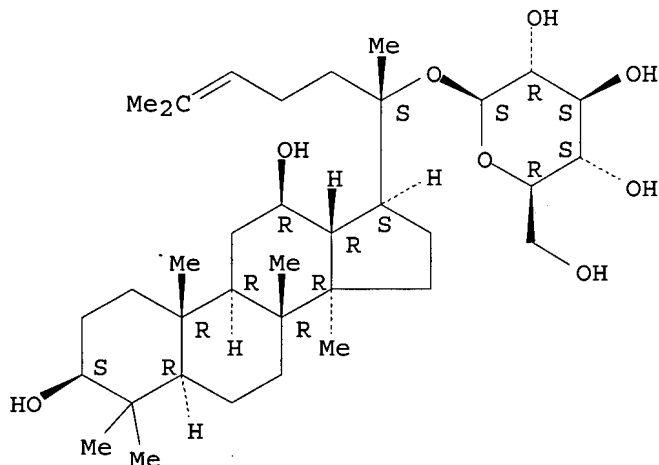
IT 39262-14-1, 20-O- β -D-Glucopyranosyl-20(S)-protopanaxadiol
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(compound K induces expression of hyaluronan synthase 2 gene in transformed human keratinocytes and increases hyaluronan in hairless mouse skin)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT:

32

THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2007:654410 CAPLUS

DOCUMENT NUMBER: 147:133487

TITLE: Ginsenosides compound K and Rh2 inhibit tumor necrosis factor- α -induced activation of the NF- κ B and JNK pathways in human astroglial cells

AUTHOR(S): Choi, Kyungsun; Kim, Myungsun; Ryu, Jeonghee; Choi, Chulhee

CORPORATE SOURCE: Laboratory of Computational Cell Biology, Department of Brain and Bioengineering, Korea Advanced Institute of Science and Technology (KAIST), Daejeon, 305-701, S. Korea

SOURCE: Neuroscience Letters (2007), 421(1), 37-41

CODEN: NELED5; ISSN: 0304-3940

PUBLISHER: Elsevier Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Ginsenosides, the main component of Panax ginseng, have been known for the anti-inflammatory and anti-proliferative activities. In this study, we investigated the mol. mechanisms responsible for the anti-inflammatory effects of ginsenosides on activated astroglial cells. Among 13 different ginsenosides, intestinal bacterial metabolites Rh2 and compound K (C-K) showed a significant inhibitory effect on tumor necrosis factor- α (TNF- α)-induced expression of intercellular adhesion mol.-1 in human astroglial cells. Pretreatment with C-K or Rh2 suppressed TNF- α -induced phosphorylation of I κ B α kinase and the subsequent phosphorylation and degradation of I κ B α . Addnl., the same treatment inhibited TNF- α -induced phosphorylation of MKK4 and the subsequent activation of the JNK-AP-1 pathway. The inhibitory effect of ginsenosides on TNF- α -induced activation of the NF- κ B and JNK pathways was not observed in human monocytic U937 cells. These results collectively indicate that ginsenoside metabolites C-K and Rh2 exert anti-inflammatory effects by the inhibition of both NF- κ B and JNK pathways in a cell-specific manner.

IT 39262-14-1, Ginsenoside k

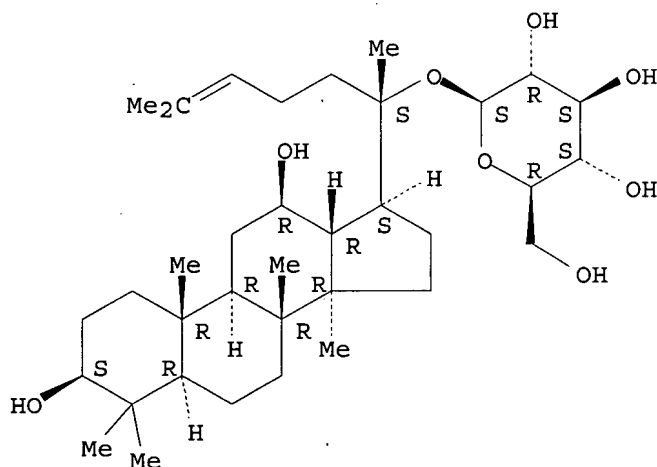
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(ginsenosides K and Rh2 inhibit TNF-induced activation of NF- κ B and JNK pathways in human astroglia: antiinflammatory effects)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2007:116233 CAPLUS

DOCUMENT NUMBER: 146:351060

TITLE: Effect of Ginsenosides on Glucose Uptake in Human
Caco-2 Cells Is Mediated through Altered Na⁺/Glucose
Cotransporter 1 Expression

AUTHOR(S): Chang, Tsu-Chung; Huang, Shu-Fen; Yang, Te-Chun; Chan,
Fang-Na; Lin, Hang-Ching; Chang, Wen-Liang

CORPORATE SOURCE: Department of Biochemistry and School of Pharmacy,
National Defense Medical Center, Taipei, Taiwan

SOURCE: Journal of Agricultural and Food Chemistry (2007),
55(5), 1993-1998

CODEN: JAFCAU; ISSN: 0021-8561

PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In this study, we measured the effect of ginsenosides on glucose uptake using the Caco-2 cell system. At submicromolar concns., these compds. exhibited marked effects on the rate of glucose transport across the differentiated Caco-2 cell monolayer. Compound K (CK), the main intestinal bacterial metabolite of the protopanaxadiol ginsenosides, significantly enhanced the steady-state glucose transport rate to about 50% of the control sample rate (from 1.54±0.09 to 2.25±0.15 nmol/min). Conversely, the protopanaxatriol ginsenoside Rg1 inhibited glucose transport to about 70% of the original rate (from 1.54±0.09 to 1.02±0.05 nmol/min). Consistent with the effect on glucose uptake rate, CK and Rg1 conferred a significant and paralleled alteration on both the protein and mRNA expression levels of the Na⁺/glucose cotransporter 1 (SGLT1) gene. Unlike SGLT1, there is no significant alteration on the protein or mRNA levels of GLUTs in CK- or Rg1-treated cells. Taken together, our results demonstrate that ginsenosides CK and Rg1 elicited potent enhancing and suppressing effects, resp., on glucose uptake across human intestinal Caco-2 monolayer through modulation of SGLT1 expression.

IT 39262-14-1

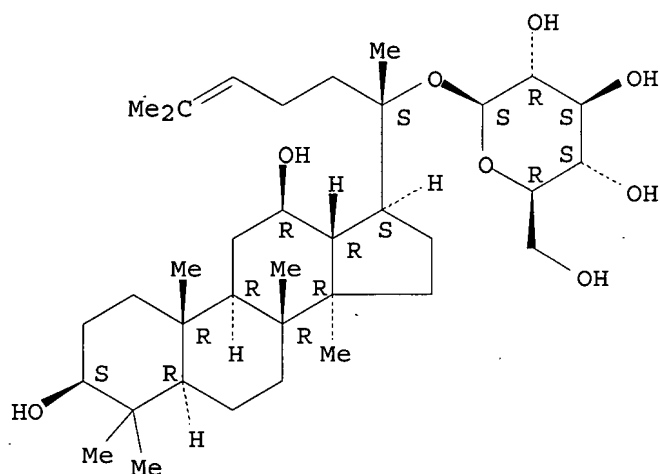
RL: DMA (Drug mechanism of action); NPO (Natural product occurrence); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)

(effect of ginsenosides on glucose uptake in human Caco-2 cells is mediated through altered Na⁺/glucose cotransporter 1 expression)

RN 39262-14-1 CAPLUS

CN β-D-Glucopyranoside, (3β,12β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:889392 CAPLUS

DOCUMENT NUMBER: 140:228662

TITLE: A novel ginseng saponin metabolite induces apoptosis and down-regulates fibroblast growth factor receptor 3 in myeloma cells

AUTHOR(S): Choi, Hyun Ho; Jong, Hyun-Soon; Park, Jung-Hyun; Choi, Seongwon; Lee, Jung Weon; Kim, Tae-You; Otsuki, Takemi; Namba, Masayoshi; Bang, Yung-Jue

CORPORATE SOURCE: National Research Laboratory for Cancer Epigenetics, Cancer Research Institute, Seoul National University College of Medicine, Chongno-Ku, Seoul, S. Korea

SOURCE: International Journal of Oncology (2003), 23(4), 1087-1093

CODEN: IJONES; ISSN: 1019-6439

PUBLISHER: International Journal of Oncology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Ginseng (the root of *Panax ginseng* C.A. Meyer, Araliaceae) has been used as a crude drug taken orally for preventive and therapeutic purposes in Asian countries as a traditional medicine. In the current study, we have investigated the antitumor effect of a novel ginseng protopanaxadiol saponin bacterial metabolic derivative, 20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol (IH-901), in eight human myeloma cell lines. IH-901 inhibited the proliferation of all myeloma cell lines examined. Despite the fibroblast growth factor receptor 3 (FGFR3) overexpression due to a chromosomal translocation t(4;14)(q16.3;q32.3) in KMS-11 myeloma cells, IH-901 induced apoptosis in a dose- and time-dependent way in this cell line. Treatment of KMS-11 with IH-901 resulted in the formation of internucleosomal DNA fragments, poly (ADP-ribose) polymerase cleavage, and the activation of caspase-3. IH-901 also caused the down-regulation of FGFR3 mRNA and protein expression and inhibited ERK activity in KMS-11 cells. Our results demonstrate that IH-901 induces apoptosis and inhibits FGFR3 expression and signaling in KMS-11 cells, suggesting candidacy for the chemoprevention and the treatment of myeloma.

IT 39262-14-1, IH-901

RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

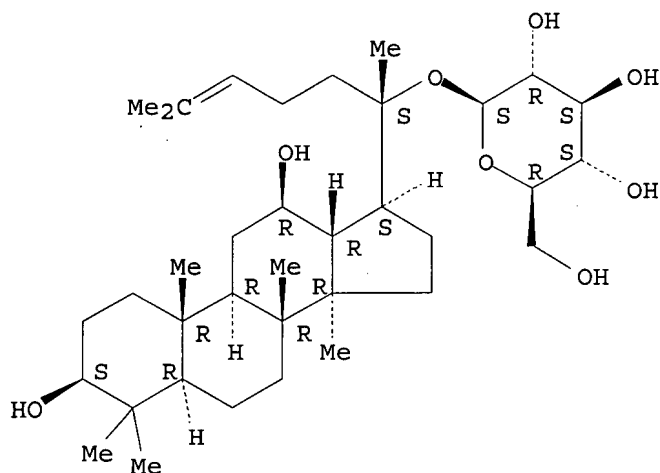
(ginseng saponin metabolite induction of apoptosis and down-regulation of FGF receptor 3 in human myeloma cells)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-

yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1998:355560 CAPLUS

DOCUMENT NUMBER: 129:117479

TITLE: An intestinal bacterial metabolite of ginseng protopanaxadiol saponins has the ability to induce apoptosis in tumor cells

AUTHOR(S): Wakabayashi, Chisato; Murakami, Koji; Hasegawa, Hideo; Murata, Jun; Saiki, Ikuo

CORPORATE SOURCE: Department of Pathogenic Biochemistry, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan

SOURCE: Biochemical and Biophysical Research Communications (1998), 246(3), 725-730

CODEN: BBRCA9; ISSN: 0006-291X

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

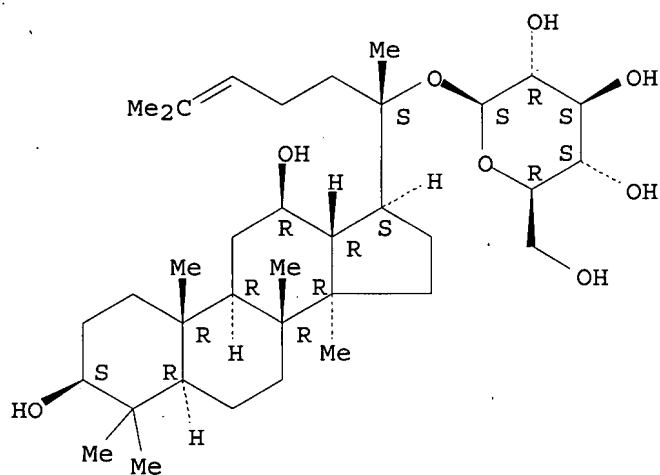
AB Our previous study demonstrated that the in vivo anti-metastatic effect induced by oral administration of ginseng protopanaxadiol saponins was mediated by their metabolic component M1, and that the growth, invasion and migration of tumor cells were inhibited by M1 but not by ginsenosides. Here we investigated the inhibitory mechanism of M1 on the growth of tumor cells. M1 inhibited the proliferation of B16-BL6 mouse melanoma cells in a time- and dose-dependent manner, with accompanying morphol. changes at the concentration of 20 μ M. In addition, at 40 μ M M1 induced apoptotic cell death within 24 h. Fluorescence microscopy revealed that dansyl M1 entered the cytosol and quickly reached the nuclei (approx. 15 min). Western blot anal. revealed that M1 rapidly up-regulated the expression of p27Kip1, but down-regulated the expression of c-Myc and cyclin D1 in a time-dependent manner. Thus, the regulation of apoptosis-related proteins by M1 is responsible for the induction of apoptotic cell death, and this probably leads to the anti-metastatic activity in vivo. (c) 1998 Academic Press.

IT 39262-14-1

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses) (antimetastatic activity of ginseng protopanaxadiol saponin metabolite M1)

RN 39262-14-1 CAPLUS
CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry.~ Rotation (+).



REFERENCE COUNT:

27

THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 1 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2007:771624 CAPLUS

DOCUMENT NUMBER: 147:439626

TITLE: Anti-proliferation and apoptosis induced by a novel intestinal metabolite of ginseng saponin in human hepatocellular carcinoma cells

AUTHOR(S): Ming, Yan-lin; Song, Gang; Chen, Liang-hua; Zheng, Zhi-zhong; Chen, Zhong-yan; Ouyang, Gao-liang; Tong, Qing-xuan

CORPORATE SOURCE: Key Laboratory of Ministry of Education for Cell Biology and Tumor Cell Engineering, School of Life Sciences, Xiamen University, Xiamen, 361005, Peop. Rep. China

SOURCE: Cell Biology International (2007), 31(10), 1265-1273
CODEN: CBIIEV; ISSN: 1065-6995

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB 20-O-(β -D-glucopyranosyl)-20(S)-protopanaxadiol (IH-901), a novel intestinal bacterial metabolite of ginseng protopanaxadiol saponins, is reported to induce apoptosis in a variety of cancer cells. We purified the compound and measured its in vitro anti-tumor activity. IH-901 inhibited cell growth of human hepatocellular carcinoma SMMC7721 cells in a dose- and time-dependent manner. We also found that IH-901 induced apoptotic cell death concurrent with cell cycle arrest in G0-G1 phase in SMMC7721 cells. At the mol. level, we show that IH-901 upregulates cytochrome c, p53, and Bax expression, and downregulates pro-caspase-3 and pro-caspase-9 expressions in a dose-dependent manner, while the levels of Bcl-2 and Bcl-XL were unchanged in IH-901-treated SMMC7721 cells. These results provide significant insight into the anticarcinogenic action of IH-901.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2006:1240475 CAPLUS

DOCUMENT NUMBER: 146:13180

TITLE: Pharmaceutical composition for treating arthritis comprising 20-O- β -glucopyranosyl-20(S)-protopanaxadiol capable of inhibiting expression of matrix metalloproteinase-3 and matrix metalloproteinase-13

INVENTOR(S): Song, Young Wook; Lee, Eun Young

PATENT ASSIGNEE(S): S. Korea

SOURCE: Repub. Korean Kongkae Taeho Kongbo, No pp. given
CODEN: KRXXA7

DOCUMENT TYPE: Patent

LANGUAGE: Korean

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
KR 2006067900	A	20060620	KR 2005-123633	20051215
PRIORITY APPLN. INFO.:			KR 2004-105999	A 20041215

AB A pharmaceutical composition comprising 20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol is provided to inhibit formation of NO and effectively block the activation of mitogen activated protein kinase (MAP kinase) mediating apoptosis of chondrocyte, thereby being effective for treating arthritis. The pharmaceutical composition for treating arthritis comprises a therapeutically effective amount of 20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol or a prodrug thereof as an effective ingredient, and a pharmaceutically acceptable carrier. In the composition, the prodrug is

saponin isolated from Panax ginseng such as Rb1, Rb2, Rc, Rd, Re, Rf, Rg1 and Rg3.

L13 ANSWER 3 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2006:476094 CAPLUS

DOCUMENT NUMBER: 145:410451

TITLE: Metabolite 1 of Protopanaxadiol-Type Saponins, an Axonal Regenerative Factor, Stimulates Teneurin-2 Linked by PI3-Kinase Cascade

AUTHOR(S): Tohda, Chihiro; Hashimoto, Itsuki; Kuboyama, Tomoharu; Komatsu, Katsuko

CORPORATE SOURCE: Division of Biofunctional Evaluation, Research Center for Ethnomedicine, Institute of Natural Medicine, University of Toyama, Toyama, Japan

SOURCE: Neuropsychopharmacology (2006), 31(6), 1158-1164
CODEN: NEROEW; ISSN: 0893-133X

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We previously showed that 20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol (M1), a metabolite of protopanaxadiol-type ginseng saponins by intestinal bacteria had axonal extension activity in degenerated neurons, and improved memory disorder and synaptic loss induced by an active fragment of amyloid β , A β (25-35). It is unknown how M1 shows these effects in neurons. To clarify the signal transduction mechanism of M1-induced axonal extension, phosphorylated proteins by M1 stimulation were identified because most cellular signal pathways are regulated by phosphorylation/dephosphorylation. The combination of immunopptn. and MALDI-TOF-MS revealed that teneurin-2 and mPar3 were specifically phosphorylated by M1 stimulation. Because mPar3 is known as an axonal specifying mol. and to be regulated by phosphatidylinositol 3-kinase (PI3-kinase), the involvement of teneurin-2 and PI3-kinase in the M1 signal was studied. In teneurin-2-deficient cortical neurons, M1-induced axonal extension and PI3-kinase activation were significantly inhibited. In addition, treatment with PI3-kinase inhibitor also reduced M1-induced axonal extension. These results suggest that M1 induces axonal outgrowth through the teneurin-2-PI3-kinase cascade.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 4 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:607823 CAPLUS

DOCUMENT NUMBER: 143:318544

TITLE: G1 phase arrest of the cell cycle by a ginseng metabolite, compound K, in U937 human monocytic leukemia cells

AUTHOR(S): Kang, Kyoung Ah; Kim, Yeong Wan; Kim, Seung Uk; Chae, Sungwook; Koh, Young Sang; Kim, Hee Sun; Choo, Min Kyung; Kim, Dong Hyun; Hyun, Jin Won

CORPORATE SOURCE: Department of Biochemistry, College of Medicine and Applied Radiological Science Research Institute, Cheju National University, Jeju-si, 690-756, S. Korea

SOURCE: Archives of Pharmacal Research (2005), 28(6), 685-690
CODEN: APHRDQ; ISSN: 0253-6269

PUBLISHER: Pharmaceutical Society of Korea

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We recently reported that the ginseng saponin metabolite, compound K (20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol, IH901), inhibits the growth of U937 cells through caspase-dependent apoptosis pathway. In this study, we further characterized the effects of compound K on U937 cells and found that, in addition to apoptosis, compound K induced the arrest of the G1 phase. The compound K treated U937 cells showed increased p21

expression; an inhibitory protein of cyclin-cdk complex. The up-regulation of p21 was followed by the inactivation of cyclin D and the cdk4 protein, which act at the early G1 phase, and cyclin E, which acts at the late G1 phase. Furthermore, compound K induced the activation of JNK and the transcription factor AP-1, which is a downstream target of JNK. These findings suggest that the up-regulation of p21 and activation of JNK in the compound K treated cells contribute to the arrest of the G1 phase.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 5 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:485182 CAPLUS

DOCUMENT NUMBER: 143:259627

TITLE: Induction of apoptosis by ginseng saponin metabolite in U937 human monocytic leukemia cells

AUTHOR(S): Kang, Kyoung A. H.; Lim, Hee Kyoung; Kim, Seung U. K.; Kim, Yeong Wan; Kim, Won Taek; Chung, H. A. Sook; Choo, Min Kyung; Kim, Dong Hyun; Kim, Hee Sun; Shim, M. I. J. A.; Chung, Myung-Hee; Hyun, Jin Won

CORPORATE SOURCE: Department of Biochemistry College of Medicine, Cheju National University, Jeju, 690-756, S. Korea

SOURCE: Journal of Food Biochemistry (2005), 29(1), 27-40
CODEN: JFBIDW; ISSN: 0145-8884

PUBLISHER: Blackwell Publishing, Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We report the cytotoxicity of the ginseng saponin metabolite, Compound K (20-O-D-glucopyranosyl-20(S)-protopanaxadiol, IH901) on various human leukemia cell lines. Compound K had the most effect on U937, a human monocytic leukemia cell line among the tested cell lines. Compound K-treated U937 cells showed characteristics of apoptosis: an exposure of phosphatidylserine from the inner cell membrane to the outer cell membrane, the formation of apoptotic bodies and DNA fragmentation. Compound K induced apoptosis by up-regulating Bax, disrupting the mitochondrial membrane potential, and by activating caspase 9 and caspase 3. Therefore, we suggest that Compound K inhibits U937 cell growth by inducing apoptosis through the up-regulation of Bax and caspase activation.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 6 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:452307 CAPLUS

DOCUMENT NUMBER: 143:166241

TITLE: Inhibitory effect of ginsenoside Rb1 and compound K on NO and prostaglandin E2 biosyntheses of RAW264.7 cells induced by lipopolysaccharide

AUTHOR(S): Park, Eun-Kyung; Shin, Yong-Wook; Lee, Hae-Ung; Kim, Sung-Soo; Lee, Young-Churl; Lee, Boo-Yong; Kim, Dong-Hyun

CORPORATE SOURCE: College of Pharmacy, Kyung Hee University, Seoul, 130-701, S. Korea

SOURCE: Biological & Pharmaceutical Bulletin (2005), 28(4), 652-656
CODEN: BPBLEO; ISSN: 0918-6158

PUBLISHER: Pharmaceutical Society of Japan

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In this study, the antiinflammatory activities of ginsenoside Rb1, which is a main constituent of the root of Panax ginseng (Araliaceae), and of its metabolite compound K, as produced by human intestinal bacteria, on lipopolysaccharide (LPS)-induced RAW264.7 cells were investigated. Compound K potently inhibited the production of NO and prostaglandin E2 in LPS-induced RAW 264.7 cells, with IC50 values of 0.012 and 0.004 mM, resp. Compound K also reduced the expression levels of the inducible NO synthase

(iNOS) and COX-2 proteins and inhibited the activation of NF-kB, a nuclear transcription factor. Compound K inhibited the NO level produced by iNOS enzyme activity in a cell-free system, but did not inhibit COX-1 and 2 activities. When ginsenoside Rb1 was orally administered to rats, compound K, but not ginsenoside Rb1, were excreted in their urine. These findings suggest that ginsenoside Rb1 can be transformed to compound K by intestinal bacteria, and compound K may be effective against inflammation.

REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:207052 CAPLUS

DOCUMENT NUMBER: 142:273636

TITLE: Cyclooxygenase-2 inhibits novel ginseng metabolite-mediated apoptosis

AUTHOR(S): Yim, Hyung Woo; Jong, Hyun-Soon; Kim, Tai Young; Choi, Hyun Ho; Kim, Sang Gyun; Song, Sang Hyun; Kim, Juyong; Ko, Seong-Gyu; Lee, Jung Weon; Kim, Tae-You; Bang, Yung-Jue

CORPORATE SOURCE: National Research Laboratory for Cancer Epigenetics, Cancer Research Institute, Seoul National University College of Medicine, Seoul, 110-744, S. Korea

SOURCE: Cancer Research (2005), 65(5), 1952-1960
CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Recently, a novel intestinal bacterial metabolite of ginseng protopanaxadiol saponins, i.e., 20-O-(β -D-glucopyranosyl)-20(S)-protopanaxadiol (IH-901), has been reported to induce apoptosis in a variety of cancer cells. Here we show a differential effect of IH-901 on several cell types. Exposure to IH-901 for 48 h at a supposedly subapoptotic concentration of 40 μ mol/L led to both apoptotic cell death and G1 arrest in Hep3B cells, but only resulted in G1 arrest in MDA-MB-231, Hs578T, and MKN28 cells. Addnl., the treatment of MDA-MB-231, but not of Hep3B, with IH-901 up-regulated cyclooxygenase-2 (COX-2) mRNA (2 h) and protein (6 h), and enhanced the production of prostaglandin E2. In MDA-MB-231 cells, IH-901 induced the sustained activation of extracellular signal-regulated kinase (ERK), whereas inhibition of mitogen-activated protein/ERK kinase blocked IH-901-mediated COX-2 induction and resulted in apoptosis, suggesting the involvement of an ERK-COX-2 pathway. Combined treatment with IH-901 and nonsteroidal anti-inflammatory drugs inhibited COX-2 enzyme and induced apoptosis in MDA-MB-231 and Hs578T cells. Adenovirus-mediated COX-2 small interfering RNAs also effectively inhibited COX-2 protein expression and enhanced IH-901-mediated apoptosis without inhibiting ERK 1/2 phosphorylation, thus providing direct evidence that COX-2 is an antiapoptotic mol. Moreover, IH-901-mediated G1 arrest resulted from an increase in p27Kip1 mRNA and protein expression followed by a decrease in CDK2 kinase activity that was concurrent with the hypophosphorylation of Rb and p130. In conclusion, IH-901 induced both G1 arrest and apoptosis, and this apoptosis could be inhibited by COX-2 induction.

REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 8 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:1051445 CAPLUS

DOCUMENT NUMBER: 142:148545

TITLE: Changes of [3H]MK-801, [3H]muscimol and [3H]flunitrazepam Binding in Rat Brain by the Prolonged Ventricular Infusion of Transformed Ginsenosides

AUTHOR(S): Jang, Soyong; Ryu, Jong Hoon; Kim, Dong-Hyun; Oh, Seikwan

CORPORATE SOURCE: Department of Neuroscience, College of Medicine, Ewha University, Seoul, S. Korea
SOURCE: Neurochemical Research (2004), 29(12), 2257-2266
CODEN: NEREDZ; ISSN: 0364-3190
PUBLISHER: Springer Science+Business Media, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Ameliorating effects of ginseng were observed on neuronal cell death associated with ischemia or glutamate toxicity. Ginseng saponins are transformed by intestinal microflora and the transformants would be absorbed from intestine. In the present study, we have investigated the effects of transformed ginsenoside Rg3, Rh2 and compound K on the modulation of NMDA receptor and GABAA receptor binding in rat brain. The NMDA receptor binding was analyzed by quant. autoradiog. using [3H]MK-801 binding, and GABAA receptor bindings were analyzed by using [3H]muscimol and [3H]flunitrazepam binding in rat brain slices. Ginsenoside Rg3, Rh2 and compound K were infused (10 µg/10 µl/h) into rat brain lateral ventricle for 7 days, through pre-implanted cannula by osmotic minipumps (Alzet, model 2ML). The levels of [3H]MK-801 binding were highly decreased in almost all regions of frontal cortex and hippocampus by ginsenoside Rh2 and compound K. The levels of [3H]muscimol binding were elevated in part of frontal cortex and granule layer of cerebellum by the treatment of ginsenoside Rh2 and compound K. However, the [3H]flunitrazepam binding was not modulated by any tested ginsenosides. Ginsenoside Rh2 and compound K induced the downregulation of the [3H]MK-801 binding as well as upregulation of the and [3H]muscimol binding in a region-specific manner after prolonged infusion into lateral ventricle. However, ginsenoside Rg3 did not show the significant changes of ligand bindings. In addition, ginsenoside Rh2 decreased the expression of nNOS in the hippocampus although Rg3 decreased the expression in the cortex. These results suggest that biotransformed ginsenoside Rh2 and compound K could play an important role in the biol. activities in the central nervous systems and neurodegenerative disease.

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 9 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:317557 CAPLUS

DOCUMENT NUMBER: 141:374612

TITLE: Aβ(25-35)-Induced Memory Impairment, Axonal Atrophy, and Synaptic Loss are Ameliorated by M1, A Metabolite of Protopanaxadiol-Type Saponins

AUTHOR(S): Tohda, Chihiro; Matsumoto, Noriaki; Zou, Kun; Meselhy, Meselhy R.; Komatsu, Katsuko

CORPORATE SOURCE: Institute of Natural Medicine, Research Center for Ethnomedicines, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan

SOURCE: Neuropsychopharmacology (2004), 29(5), 860-868
CODEN: NEROEW; ISSN: 0893-133X

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We previously screened neurite outgrowth activities of several Ginseng drugs in human neuroblastoma, and demonstrated that protopanaxadiol (ppd)-type saponins were active constituents. Since ppd-type saponins are known to be completely metabolized to 20-O-β-D-glucopyranosyl-20(S)-protopanaxadiol (M1) by intestinal bacteria when taken orally, M1 and ginsenoside Rb1, as a representative of ppd-type saponins, were examined for cognitive disorder. In a mouse model of Alzheimer's disease (AD) by Aβ(25-35) i.c.v. injection, impaired spatial memory was recovered by p.o. administration of ginsenoside Rb1 or M1. Although the expression levels of phosphorylated NF-H and synaptophysin were reduced in the cerebral cortex and the hippocampus of Aβ(25-35)-injected mice, their levels in ginsenoside Rb1- and M1-treated mice were

almost completely recovered up to control levels. Potencies of the effects were not different between ginsenoside Rb1 and M1 when given orally, suggesting that most of the ginsenoside Rb1 may be metabolized to M1, and M1 is an active principal of ppd-type saponins for the memory improvement. In cultured rat cortical neurons, M1 showed extension activity of axons, but not dendrites. The axon-specific outgrowth was seen even when neuritic atrophy had already progressed in response to administration of A β (25-35) as well as in the normal condition. These results suggest that M1 has axonal extension activity in degenerated neurons, and improve memory disorder and synaptic loss induced by A β (25-35). M1 was shown to be effective in vitro and in vivo, indicating that Ginseng drugs containing ppd-type saponins may reactivate neuronal function in AD by p.o. administration.

REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:553077 CAPLUS
DOCUMENT NUMBER: 140:8435
TITLE: Anti-wrinkle activity of hydrolyzed ginseng saponins
AUTHOR(S): Yeom, M. H.; Sung, D. S.; Woo, K. S.; Kang, B. Y.; Kim, D. H.; Chang, I. S.; Kang, H. H.; Lee, O. S.
CORPORATE SOURCE: Pacific R+D Center, Kyunggi-do, S. Korea
SOURCE: Cosmetics & Toiletries (2003), 118(3), 77-80, 82
CODEN: CTOIDG; ISSN: 0361-4387
PUBLISHER: Allured Publishing Corp.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Hydrolyzed ginseng saponins, produced by enzymic hydrolysis, increase collagen synthesis and decrease the expression of MMP-1. Also, the formation of nano-emulsions containing hydrolyzed ginseng saponins significantly increases the collagen synthesis.

REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2000:526438 CAPLUS
DOCUMENT NUMBER: 133:246874
TITLE: Induction of apoptosis by a novel intestinal metabolite of ginseng saponin via cytochrome c-mediated activation of caspase-3 protease
AUTHOR(S): Lee, S.-J.; Ko, W.-G.; Kim, J.-H.; Sung, J.-H.; Lee, S.-J.; Moon, C.-K.; Lee, B.-H.
CORPORATE SOURCE: College of Pharmacy and Medicinal Resources Research Center, Wonkwang University, Iksan, Chonbuk, 570-749, S. Korea
SOURCE: Biochemical Pharmacology (2000), 60(5), 677-685
CODEN: BCPA6; ISSN: 0006-2952
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Ginseng saponins exert various important pharmacol. effects with regard to the control of many diseases including cancer. The novel intestinal bacterial metabolites of ginseng protopanaxadiol saponins have recently been found and isolated after the oral administration of ginseng extract in human and rats. 20-O-(β -d-Glucopyranosyl)-20(S)-protopanaxadiol (IH-901) formed from ginsenosides Rb1, Rb2, and Rc is of particular interest in cancer chemoprevention and treatment. We investigated the effects of IH-901 on the human myeloid leukemia cell line HL-60 in terms of inhibition of proliferation and induction of apoptosis. IH-901 showed a significant cytotoxic activity in HL-60 cells (IC₅₀ = 24.3 μ M) following a 96-h incubation. Treatment of HL-60 cells with IH-901 resulted in the formation of internucleosomal DNA fragments. The dose- and time-dependent induction of apoptosis by IH-901 was demonstrated in

sandwich enzyme immunoassay and the results were confirmed by flow cytometric anal. Morphol. examination of IH-901-treated samples showed cells with chromatin condensation, cell shrinkage, and nuclear fragmentation, all typical characteristics of apoptotic cells. The treatment of HL-60 cells with IH-901 caused activation of caspase-3 protease and subsequent proteolytic cleavage of poly(ADP-ribose) polymerase. IH-901 did not affect the expression of antiapoptotic protein Bcl-2 but did cause a release of mitochondrial cytochrome c into cytosol. In conclusion, our results demonstrate that IH-901 dramatically suppresses HL-60 cell growth by inducing programmed cell death through activation of caspase-3 protease, which occurs via mitochondrial cytochrome c release independently of Bcl-2 modulation. These results may provide a pivotal mechanism for the use of IH-901 in the prevention and treatment of leukemia.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1998:354596 CAPLUS

DOCUMENT NUMBER: 129:76147

TITLE: Expression of in vivo anti-metastatic effect of ginseng protopanaxatriol saponins is mediated by their intestinal bacterial metabolites after oral administration

AUTHOR(S): Wakabayashi, Chisato; Hasegawa, Hideo; Murata, Jun; Ichiyama, Masamori; Saiki, Ikuro

CORPORATE SOURCE: Departments of Pathogenic Biochemistry, Research Institute for Wakan-Yaku, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan

SOURCE: Wakan Iyakugaku Zasshi (1997), 14(4), 288-289
CODEN: WIZAE; ISSN: 1340-6302

PUBLISHER: Wakan Iyaku Gakkai

DOCUMENT TYPE: Journal

LANGUAGE: Japanese

AB The antimetastatic effect of ginseng protopanaxatriol saponins was studied in mice transplanted with melanoma cells. following oral administration. The results indicated that the antimetastatic effect of ginseng protopanaxatriol saponins is mediated by their intestinal bacterial metabolites after oral administration.

L13 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1995:758270 CAPLUS

DOCUMENT NUMBER: 123:188022

TITLE: Immunomodulating activity of tetracyclic triterpene glycosides of the dammaran and holostan series

AUTHOR(S): Popov, A. M.; Atopkina, L. N.; Samoshina, N. F.; Uvarova, N. I.

CORPORATE SOURCE: Far East Department Russian Academy Sciences, Vladivostok, Pacific Ocean Institute Bioorganic Chemistry, Russia

SOURCE: Antibiotiki i Khimioterapiya (1994), 39(9-10), 19-25
CODEN: ANKHEW; ISSN: 0235-2990

PUBLISHER: Meditsina

DOCUMENT TYPE: Journal

LANGUAGE: Russian

AB Immunomodulating activity of triterpene glycosides of the holostan series (holothurins A and B, holothurin A2 and holotoxin A1) and triterpene glycosides of the dammaran series (3-O-monoglycoside, 12-O-monoglycoside and 20-O-monoglycoside of protopanaxadiol and 3-O-monoglycoside of betulafolientriol) was studied in vitro. In low concns. the triterpene glycosides showed mitogenic activity and modulated the immune response. The similarity in the action of the glycosides was first of all observed with respect to the dose-dependent duality of their effects i.e. the diametrically opposite action of the high and low doses. The

expression of the effects was likely determined by the chemical structure of the triterpene glycosides. Liberation of the soluble mediators served as a secondary signal to the clonal expansion and differentiation of the cells.

ACCESSION NUMBER: 2005:1149653 CAPLUS
 DOCUMENT NUMBER: 143:410642
 TITLE: Ginsenoside-encapsulated polymer microcapsules for cosmetics, and manufacture thereof
 INVENTOR(S): Lee, Young Suk; Lee, Sang Il; Kim, Jin Won; Kim, Ju No; Jang, Lee Sup
 PATENT ASSIGNEE(S): Amorepacific Corporation, S. Korea
 SOURCE: Jpn. Kokai Tokkyo Koho, 16 pp.
 CODEN: JKXXAF
 DOCUMENT TYPE: Patent
 LANGUAGE: Japanese
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2005298510	A	20051027	JP 2005-114681	20050412
KR 2005100471	A	20051019	KR 2004-25710	20040414
PRIORITY APPLN. INFO.:			KR 2004-25710	A 20040414

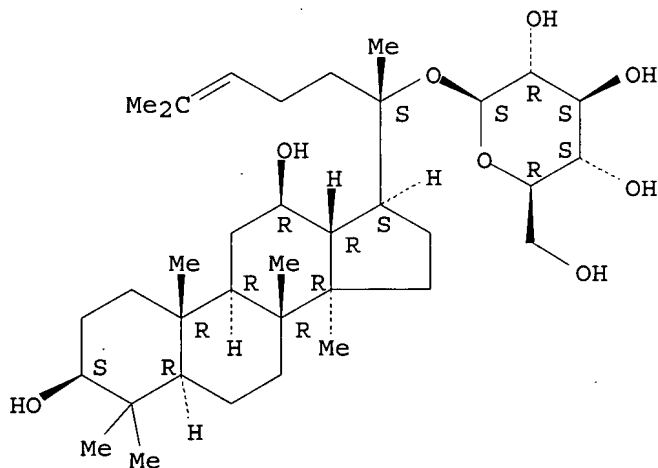
AB The invention relates to polymer microcapsules containing ginsenoside, suitable for use in an anti-aging cosmetic composition, wherein the use of the microcapsules prevents discoloration the cosmetic composition and malodor due to ginsenoside during storage. A method for manufacturing the microcapsules is also disclosed. For example, polycaprolactone microcapsules containing Panax ginseng-derived ginsenoside were prepared in the presence of polyvinyl alc. dispersion stabilizer. The obtained microcapsules 5 parts was mixed with other ingredients to obtain a skin-softening lotion.

IT 39262-14-1
 RL: COS (Cosmetic use); NPO (Natural product occurrence); PAC (Pharmacological activity); PEP (Physical, engineering or chemical process); PYP (Physical process); BIOL (Biological study); OCCU (Occurrence); PROC (Process); USES (Uses)
 (ginsenoside-encapsulated polymer microcapsules for cosmetics, and manufacture thereof)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



ACCESSION NUMBER: 2003:673814 CAPLUS
 DOCUMENT NUMBER: 139:202445
 TITLE: Bioconversion of ginseng components for therapeutic uses
 INVENTOR(S): Lee, Ok Don; Son, Fan Zon; Kim, Hyon Doson; Be, A. Un; Han, Zu Myon; Chu, Gyon Min; Bak, Gyon Un
 PATENT ASSIGNEE(S): Ilha K. K., S. Korea
 SOURCE: Jpn. Kokai Tokkyo Koho, 13 pp.
 CODEN: JKXXAF
 DOCUMENT TYPE: Patent
 LANGUAGE: Japanese
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2003238424	A	20030827	JP 2003-10301	20030117
KR 2003065273	A	20030806	KR 2002-39967	20020710
CN 1435179	A	20030813	CN 2003-101549	20030115
HK 1058147	A1	20060825	HK 2004-100932	20040212
PRIORITY APPLN. INFO.:			KR 2002-5369	A 20020130
			KR 2002-39967	A 20020710

AB This invention relates to ginseng compns. bioconverted by lactic acid bacteria or enterobacteria. The compns. comprise the ratio of (20-O- β -D-glucopyranosyl-20S-protopanaxadiol plus ginsenoside F2) to (ginsenoside Rc plus ginsenoside Rd plus ginsenoside Rb1 plus ginsenoside Rb2) being ≥ 0.1 . The compns. are obtained by (1) suspending ginseng (or exts.) raw materials in water, (2) adding lactic acid bacteria or enterobacteria for cultivation, (3) isolating the bioconverted ginseng solution by concentrating, freeze-drying, drying, and/or centrifuging. The compns.

are used for prevention of allergies, aging, and tumors. Also, beverages are formulated containing the compns.

IT 39262-14-1

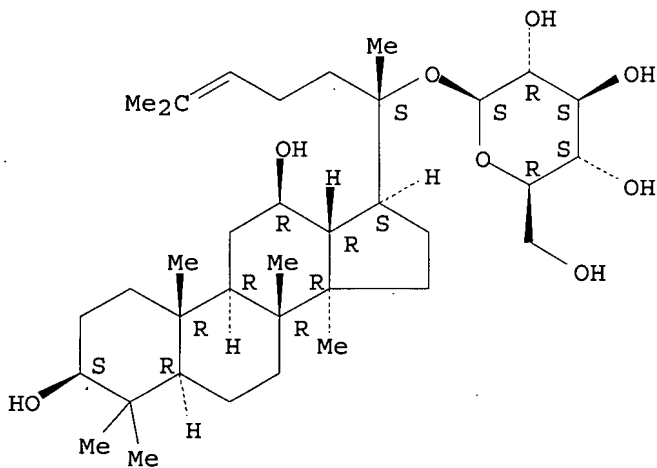
RL: NPO (Natural product occurrence); PAC (Pharmacological activity); BIOL (Biological study); OCCU (Occurrence)

(bioconversion of ginseng components for therapeutic uses)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

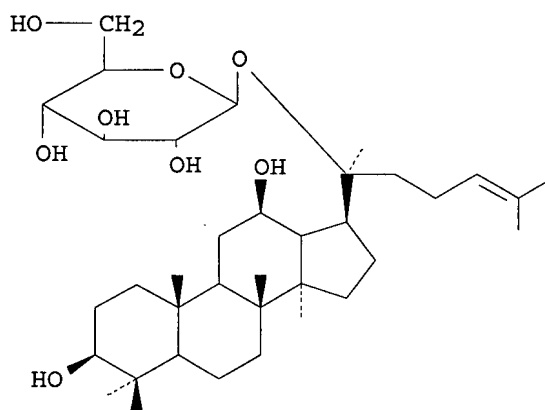
Absolute stereochemistry. Rotation (+).



ACCESSION NUMBER: 2003:550072 CAPLUS
 DOCUMENT NUMBER: 139:122444
 TITLE: Nanoemulsion comprising metabolites of ginseng saponin and a skin-care composition for anti-aging
 INVENTOR(S): Yoo, Byung Hee; Kang, Byung Young; Yeom, Myeong Hoon; Sung, Dae Seok; Ju, Hee Kyung; Han, Sang Hoon; Kim, Han Kon
 PATENT ASSIGNEE(S): Pacific Corporation, S. Korea
 SOURCE: Eur. Pat. Appl., 32 pp.
 CODEN: EPXXDW
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1327434	A1	20030716	EP 2003-290014	20030103
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
KR 2003060017	A	20030712	KR 2002-613	20020105
KR 2003060018	A	20030712	KR 2002-614	20020105
KR 2003080429	A	20031017	KR 2002-19032	20020408
KR 2003094523	A	20031218	KR 2002-29179	20020527
JP 2003212776	A	20030730	JP 2002-374691	20021225
US 2003175315	A1	20030918	US 2003-336024	20030103
US 2006216261	A1	20060928	US 2006-443271	20060531
PRIORITY APPLN. INFO.:			KR 2002-613	A 20020105
			KR 2002-614	A 20020105
			KR 2002-19032	A 20020408
			KR 2002-29179	A 20020527
			US 2003-336024	A3 20030103

GI



I

AB Disclosed is a nanoemulsion prepared by emulsifying main metabolites of ginseng saponins obtained by conversion of glucose, i.e. compound K (I), ginsenoside F1 (20-O- β -D-glucopyranosyl-20(S)-protopanaxatriol) and compound Y (20-O-[α -L-arabinopyranosyl(1 \rightarrow 6)- β -D-glucopyranosyl]-20(S)-protopanaxadiol); and admixt., in fine emulsion or liposome with dermatropic emulsifier by nano-emulsification; and having enhanced skin penetration, so to be effective in promoting proliferation of fibroblast and biosynthesis of collagen.
 IT 39262-14-1, 20-O- β -D-Glucopyranosyl-20(S)-protopanaxadiol

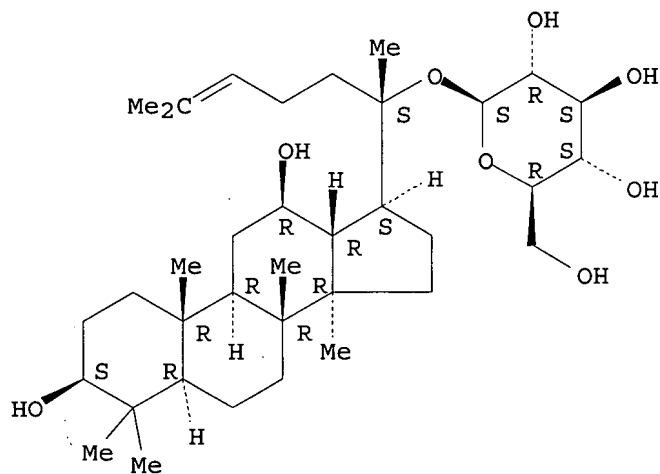
RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study);
USES (Uses)

(nanoemulsion comprising metabolites of ginseng saponin and a skin-care
composition for anti-aging)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-
yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT:

7

THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2003:651450 CAPLUS

DOCUMENT NUMBER: 140:35347

TITLE: Inhibition of intracerebroventricular injection stress-induced plasma corticosterone levels by intracerebroventricularly administered compound K, a ginseng saponin metabolite, in mice

AUTHOR(S): Kim, Do-Hoon; Jung, Jun-Sub; Moon, Yoo-Sun; Sung, Jong-Hwan; Suh, Hong-Won; Kim, Yung-Hi; Song, Dong-Keun

CORPORATE SOURCE: Departments of Psychiatry, College of Medicine, Institute of Natural Medicine, Hallym University, Chunchon, 200-702, S. Korea

SOURCE: Biological & Pharmaceutical Bulletin (2003), 26(7), 1035-1038

CODEN: BPBLEO; ISSN: 0918-6158

PUBLISHER: Pharmaceutical Society of Japan

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Effects of major intestinal metabolites of ginsenosides, including compound K (IH-901, 20-O- β -D-glucopyranosyl-20(S)-protopanaxadiol), compound Y (IH-902, 20-O-[α -L-arabinopyranosyl (1 \rightarrow 6)- β -D-glucopyranosyl]-20(S)-protopanaxadiol), and ginsenoside Mc (IH-903, 20-O-[α -L-arabinofuranosyl (1 \rightarrow 6)- β -D-glucopyranosyl]-20(S)-protopanaxadiol), on acute stress-induced plasma corticosterone levels were studied in mice. Intracerebroventricularly (i.c.v.) administered compound K (1 μ g) attenuated the i.c.v. injection stress-induced increase in plasma corticosterone level, and this inhibitory effect was not affected by co-administered NG-nitro-L-arginine Me ester, a nitric oxide synthase inhibitor. Compound K administered i.p. affected neither the i.c.v. injection stress- nor the immobilization stress-induced increase in plasma corticosterone levels. Compound K and ginsenoside Mc did not affect plasma corticosterone levels induced by the two stress modalities used in this study.

IT 39262-14-1, IH-901

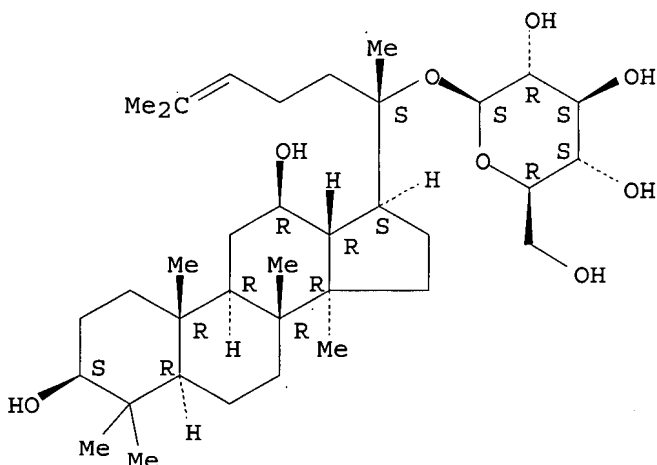
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(intracerebroventricular injection stress-induced corticosterone inhibition by ginsenosides intestinal metabolites)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT:

18

THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L27 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:555114 CAPLUS

DOCUMENT NUMBER: 140:52998

TITLE: Antiallergic activity of ginseng and its ginsenosides

AUTHOR(S): Choo, Min-Kyung; Park, Eun-Kyung; Han, Myung Joo; Kim, Dong-Hyun

CORPORATE SOURCE: College of Pharmacy, Kyung Hee University, Seoul, S. Korea

SOURCE: Planta Medica (2003), 69(6), 518-522

CODEN: PLMEAA; ISSN: 0032-0943

PUBLISHER: Georg Thieme Verlag

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In this study, we measured the antiallergic activities of ginsenosides isolated from the root of *Panax ginseng* (Araliaceae), and of their metabolites, as produced by human intestinal bacteria. Compound K, which was identified as a main metabolite, had the most potent inhibitory activity on β -hexosaminidase release from RBL-2H3 cells and on the PCA reaction. The inhibitory activity of compound K was more potent than that of disodium cromoglycate, one of the com. anti-allergic drugs. This compound demonstrated a membrane stabilizing action on differential scanning calorimetry. However, compound K did not inhibit the activation of hyaluronidase and did not scavenge active oxygen. These results suggest that the antiallergic action of compound K originates from its cell membrane stabilizing activity and that the ginsenosides of ginseng are prodrugs with extensive antiallergic properties.

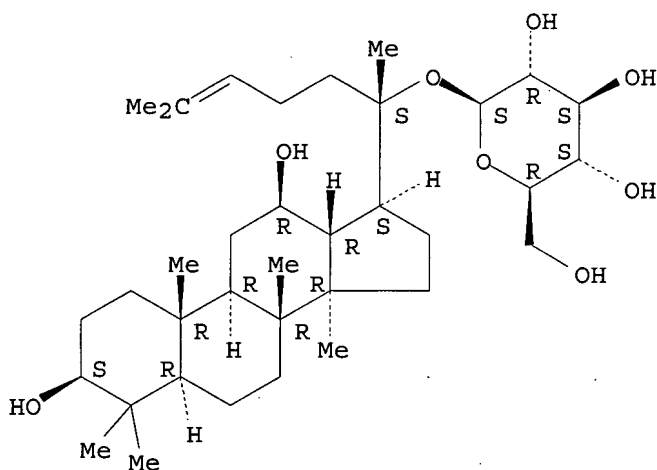
IT 39262-14-1, Protopanaxadiol 20-O-glucoside

RL: NPO (Natural product occurrence); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses) (antiallergic activity of ginseng and its ginsenosides)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT:

23

THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2006:1099507 CAPLUS

DOCUMENT NUMBER: 145:419427

TITLE: Dicarboxylic acid ester derivatives of ginsenoside, pharmaceutical preparations containing the same, and preparation thereof

INVENTOR(S): Chen, Hui-Ling; Huang, Ying-Ming; Chang, Ching-Te; Chuang, Wen-Yi

PATENT ASSIGNEE(S): Amersin Bioscience International, Inc., Taiwan

SOURCE: U.S. Pat. Appl. Publ., 23pp.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2006234956	A1	20061019	US 2006-404303	20060414
WO 2006113495	A2	20061026	WO 2006-US14191	20060414
WO 2006113495	A3	20070607		
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW			
RW:	AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA			

PRIORITY APPLN. INFO.: US 2005-671878P P 20050415

OTHER SOURCE(S): CASREACT 145:419427; MARPAT 145:419427

AB The present invention relates to a series of dicarboxylic acid ester derivs. of ginsenosides, such as succinate and glutarate derivs. of 20-O- β -D-glucopyranosyl-protopanaxadiol (compound K, abbreviated as CK), preparation thereof and pharmaceutical uses thereof. The dicarboxylic acid ester derivs. of ginsenosides of the present invention can be used to form pharmaceutical acceptable salts thereof having a high water solubility or can be directly dissolved in an aqueous solution of metal salt, and retain the pharmaceutical activities of ginsenosides such as tumor growth inhibition and cancer preventive cytotoxicity. The dicarboxylic acid ester derivs. of ginsenosides of the present invention are thus suitable for use in the manufs. of various pharmaceutically and cosmetically acceptable dosage forms of prepsns., such as peripheral, oral, and topical dosage forms. Thus, coupling reaction of 20-O- β -D-glucopyranosyl-protopanaxadiol (CK) with succinic anhydride gave a mixture of CK succinate derivs., which were tested as antitumor agents.

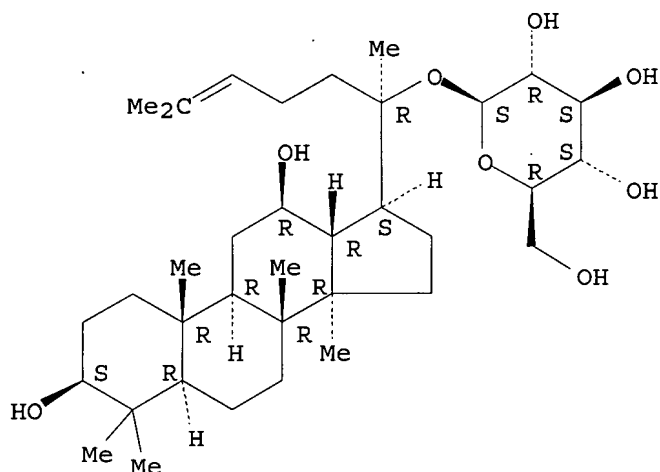
IT 183183-71-3

RL: RCT (Reactant); RACT (Reactant or reagent)
(dicarboxylic acid ester derivs. of ginsenoside, pharmaceutical prepsns. containing the same, and preparation thereof)

RN 183183-71-3 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β ,20R)-3,12-dihydroxydammar-24-en-20-yl (9CI) (CA INDEX NAME)

Absolute stereochemistry.



L30 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:531373 CAPLUS

DOCUMENT NUMBER: 141:94264

TITLE: Preparation of low polar ginsenosides and aglucons from ginsenosides by acid catalytic pyrolysis

INVENTOR(S): Ling, Yany; He, Ke-jiang; Li, Peng; Yang, Yi

PATENT ASSIGNEE(S): Dalian Institute of Chemical Physics, Peop. Rep. China

SOURCE: PCT Int. Appl., 14 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: Chinese

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004054595	A1	20040701	WO 2003-CN1055	20031211
W: JP, KR, US				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR				
CN 1508147	A	20040630	CN 2002-144780	20021213
PRIORITY APPLN. INFO.:			CN 2002-144780	A 20021213

AB Disclosure is a method for preparing low polar ginsenosides, protopanaxadiols and triols thereof by catalytic thermal decomposition of ginsenosides isolated from plants at 110-180°C under the presence of acids as catalysts. The present invention finds out that acids can work as catalysts for high temperature pyrolysis of ginsenosides, solving the problems associated with high temperature pyrolysis of natural ginsenosides in the processing technol. of dried steamed root of *Panax ginseng* (Araliaceae), and provides a handy and high effective method for preparing low polar ginsenosides. When combining with isolation and purification technol., the method provided herein can be used to prepare monomeric low polar ginsenosides in bulk. The prepared low polar ginsenosides can be used in pharmaceutical, cosmetic and functional food compns., and can also be used as starting material for preparing other bioactive compds. For example, ginsenoside 20(R)-Rg3 2,05g was prepared by thermal hydrolysis of ginsenoside diol 25g with malonic acid at 1200C for 5 h, followed by ethanol extraction

IT 39262-14-1, 20-O-β-D-Glucopyranosyl-20(S)-protopanaxadiol

RL: RCT (Reactant); RACT (Reactant or reagent)

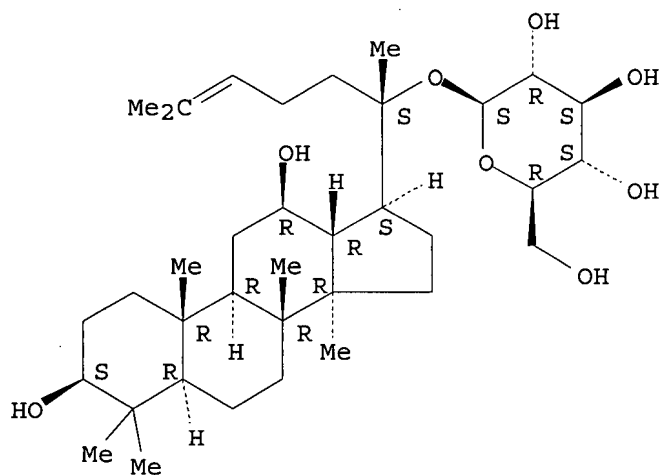
(preparation of low polar ginsenosides and aglucons from ginsenosides by acid catalytic pyrolysis)

RN 39262-14-1 CAPLUS

CN β-D-Glucopyranoside, (3β,12β)-3,12-dihydroxydammar-24-en-20-

yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT:

2

THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2005:1079048 CAPLUS

DOCUMENT NUMBER: 143:452437

TITLE: Antipruritic effect of ginsenoside Rb1 and compound K in scratching behavior mouse models

AUTHOR(S): Shin, Yong-Wook; Kim, Dong-Hyun

CORPORATE SOURCE: College of Pharmacy, Kyung Hee University, Seoul, 130-701, S. Korea

SOURCE: Journal of Pharmacological Sciences (Tokyo, Japan) (2005), 99(1), 83-88

CODEN: JPSTGJ; ISSN: 1347-8613

PUBLISHER: Japanese Pharmacological Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The antipruritic and vascular permeability-inhibitory effects of ginsenoside Rb1, a main component of ginseng frequently used as a traditional medicine in Asian countries, and its metabolite compound K by intestinal microflora were investigated in scratching behavior animal models induced by compound 48/80, substance P, and histamine. Ginsenoside Rb1 and compound K orally administered 1 and 6 h before the treatment of compound 48/80 showed antipruritic effect. These ginsenosides administered at a dose of 50 mg/kg 6 h before the treatment of compound 48/80 inhibited scratching behaviors by 51% and 64%, resp., compared with that of the control. These ginsenosides also inhibited the vascular permeability of skin. Compound K i.p. administered 1 h before the treatment of compound 48/80 potently inhibited the scratching behaviors induced by compound 48/80. However, i.p. administered ginsenoside Rb1 did not inhibit scratching behaviors. Compound K inhibited compound 48/80-, substance P-, and histamine-induced scratching behaviors, with 50% inhibitory doses of 4.2, 5.9, and 3.8 mg/kg, resp., and vascular permeability, with 50% inhibitory doses of 5.8, 6.8, and 4.1 mg/kg, resp. These results suggest that ginsenoside Rb1 and its metabolite compound K by intestinal microflora can improve scratching behaviors.

IT 39262-14-1

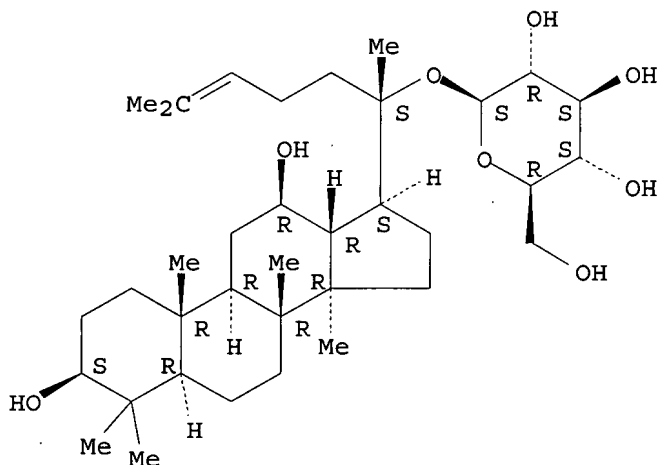
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(antipruritic and skin vascular permeability-inhibitory effect of ginsenoside Rb1 and its metabolite)

RN 39262-14-1 CAPLUS

CN β -D-Glucopyranoside, (3 β ,12 β)-3,12-dihydroxydammar-24-en-20-yl (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).



REFERENCE COUNT:

24

THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT